



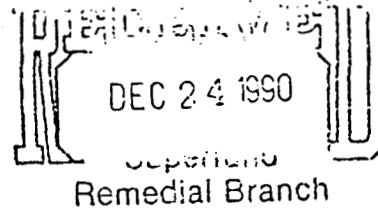
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION VIII

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DEC 21 1990

Ref: 8HWM-SM



MEMORANDUM

TO: Bonita Lavelle
Federal Facilities

FROM: Jim LaVelle, Toxicologist *llh*
Superfund Technical Section

SUBJECT: Review of Remedy Report for OU3 - SWMU 199

I have reviewed the above-referenced document and have several comments. Though parts of the document are sufficient for the stated purpose, there are a number of instances where the text is inadequate and ingenuous. Instead of providing comments on a page-by-page basis, I am writing general comments which address the basis for major conclusions in the document. This seems appropriate since deficiencies tend to permeate the text in many different sections. Also, all of the comments are based on the assumption that the data are indeed, as the authors claim, of insufficient quality to be used for a quantitative risk assessment. Since I have not at this time seen the data, I cannot evaluate this conclusion.

First, the pathways analysis is simplistic and shows little critical thinking. For example, all ingestion pathways are discounted relative to inhalation purely on the basis of low GI absorption. This is a serious error. In many, if not all, instances I have encountered in Region VIII, intake via ingestion has exceeded intake via inhalation by up to several orders of magnitude. In fact, it appears, from recent monitoring data at the Rocky Flats plant, that ingestion vs inhalation intake ratios may be on the order of 10,000:1 to 100,000:1, using standard exposure assumptions and assuming that all airborne activity is associated with respirable particles. Once particle size/radioactivity associations are known, these ratios may go even higher. Certainly, this difference in intake rates could potentially offset the difference in absorbed doses estimated between ingestion and inhalation exposures. Without quantitative data on relative intake, it is not possible to estimate relative risks due to these exposure routes, even on a qualitative basis. It is certainly possible that ingestion of contaminated soil could pose a significant risk relative to dust inhalation in the SWMU 199 area.

On a similar tack, the report suggests that Pu vs Am ratios might be on the order of 5:1 in contaminated soil. Since the

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relative absorption of Am is 10 times greater than that estimated for Pu ($1E-03$ vs $1E-04$ for most common isotopes respectively), the relative absorbed dose that would be estimated following ingestion of contaminated is 2:1 in favor of Am. Thus, discounting Am concentrations may seriously underestimate total exposure to alpha emitters. In light of the above arguments, it may be critical to monitor Am as well as Pu in the soils. It seems clear that statements such as "Plutonium is the only significant contaminant at SWMU 199..." may be incorrect. (It should be noted that the absorption factor for Pu listed in the document is incorrect. The HEAST tables list $1E-04$, not $1E-05$.)

Some statements, such as "Since plutonium has no known biological function, it can only be passively incorporated into organisms...." indicate a lack of biologic/physiologic expertise among the authors of the document. Many metals (e.g., lead, cadmium, strontium) which have no known function may be taken up via an active or facilitated transport system. I have seen no data that clearly indicates that no active uptake of plutonium takes place (e.g., via the calcium transport systems). Although it is possible that only passive uptake occurs, this needs specific documentation, not merely an ingenuous statement such as that above. Perhaps because of a lack of general physiological expertise, all of the discussion on absorption of Pu following ingestion is simplistic. For example, effects of nutritional deficits (e.g., iron) will lead to increased uptake. (Note that iron deficiency also increases uptake of lead, calcium and strontium). Further, there is evidence that absorption in young animals (and perhaps children, by extrapolation) may absorb Pu as much as 10X more efficiently than older children and adults. It may be that exposure early in life, when children also consume more soil, will contribute significantly to lifetime dose.

The document fails to cite at least one major reference on the transport of radionuclides through agriculture (Baes et al. 1984). This review and analysis is more recent than those cited and should be read before conclusions on the uptake of Pu and Am into crops are reached. It is of some interest to note that several metals which exist in extremely insoluble forms in soils (e.g., lead and cadmium) are still taken up to a significant extent into root crops. Moreover, Region VIII has some indication that the data from the Baes study may be useful when extrapolated to common Western soils. There was good agreement between an *in situ* garden study in the Salt Lake Valley and predictions made using the Baes report, both for air deposition and root uptake. I would like to see some quantitative discussion of potential exposure via this pathway before any conclusions on its significance are reached.

It is not clear that the authors of the document recognize the nature of a no-action scenario. There is no indication in the text that the probabilities of different land uses in the

foreseeable future were considered. From the EPA standpoint, it is necessary to consider a residential scenario, unless it can be shown that this is not a possible future land use. Consideration of a residential scenario would augment all of the above arguments concerning exposure pathways involving ingestion. Any risk assessment at the site should provide a careful and justifiable discussion of future land use possibilities in considering the no-action alternative.

For the inhalation pathway, the pathway-specific unit risk from inhalation of americium is the same as that for the most common plutonium isotopes. Thus, failure to consider Am as a source of alpha exposure to the lungs may ignore as much as 20% of the total. Generally EPA considers any contribution greater than 10% significant and would include such in a quantitative assessment.

The sentence "Since it has been shown that the air pathway from SWMU 199 produces negligible risk to the public, all other pathways must also produce a negligible risk." on page 49 seems clearly out-of-line given not only the above comments, but also the fact that no quantitative risk characterization (or exposure assessment) has been carried out.

The document correctly identifies a number of important data gaps which should be filled before a quantitative risk assessment is attempted. However, the recommendations were not specific enough for a critical evaluation. The new "Guidance on Data Useability for Risk Assessment" should be consulted when designing sampling and analysis plans for filling the acknowledged data gaps.

In conclusion, the document is a reasonable start at defining a conceptual site model and identifying gaps in our current information. With greater attention to basic toxicologic and physiologic concepts, it could become a useful base from which to launch a complete quantitative baseline risk assessment. I encourage you to transmit the above comments to the authors so they can be considered when the document is revised.

FCD:December 19, 1990: